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Both from the theoretical and practical standpoint, the question of the more or less complete limitation of the various cerebral functions to circumscribed regions of the cortex is one of great interest. The following cases, which have come under my observation at the Michigan Asylum for the Insane, have seemed to me worthy of mention, more as confirming observations already made than as throwing any new light on the subject.

The first case to which I will ask your attention came under my care during the first few weeks of my service. My lack of experience at the time may, perhaps, excuse, in part, the imperfection of my observation of the case.

The patient, a farmer, of French descent, aged thirty, had been under treatment nearly a year, for epilepsy, which was said to be of two years' standing at the time of his admission. His case presented no features of very special interest until January 26, 1878, when, after a very severe convulsion, he remained in a state of alarming collapse. The radial pulse was almost imperceptible, the surface was cold, and he seemed in danger of immediate dissolution. He rallied somewhat, under the administration of stimulants, but remained for three days in a stupid condition, and never regained strength sufficiently to enable him to be long out of bed.

Shortly after the attack it was observed that there was a slight lack of innervation of the right side of the face, only noticeable when the muscles were called into action, as in talking, or, more especially, in smiling. On the 11th of February, the patient, having apparently regained about his ordinary mental condition, a pretty careful examination was made, without discovering any paralysis, except as above mentioned, or any impairment of cutaneous sensibility, although he stated that he had experienced transient numbness of the hand at times. He also said that for

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several days he had hallucinations of smell—at first constant, but of late transitory. On one occasion he got up in the night, imagining that the room was full of smoke. Of late, he had noticed, at times, an odor which he compared to the vapor of alcohol, which passed away quickly, and which he thought took the place of a convulsion. I am sorry to say that no test of the sense of smell was made. I find no record of any further examination, and my recollection is that no marked change occurred in his general condition until his death, which occurred on the 28th of February, in consequence of a series of tonic convulsions, the most marked feature of which was opisthotonos, affecting, mainly, the muscles of the back.

At the autopsy, on inspection of the inferior surface of the brain, a small spot of red softening was found at the most prominent point of the left gyrus uncinatus. As I wanted a brain for dissection, it was not opened until after it had been hardened in alcohol. It was then found that a focus of red softening existed in the white matter of the anterior part of the left temporal lobe, extending to the surface, externally, as above mentioned, and internally, involving the pes hippocampi in the floor of the descending cornu of the lateral ventricle. The portion of the hippocampus major which was not discolored was swollen and softened. A very small focus of softening, without discoloration, about the size of a large pea, was also found in the white matter of the frontal lobe of the same side. No other gross lesions were found, but the perivascular spaces were very generally dilated, so as to give thin sections of the brain a worm-eaten appearance.

There seems to be good reason, both on anatomical and experimental grounds, to believe that the anterior and inferior surface of the temporal lobe has special relations to the sense of smell. Clinical evidence on that point is not, so far as I can ascertain, very abundant. Luciani and Seppilli refer to cases reported by Sander, Westphal and Schlager, none of which are accessible to me, in which anosmia and hallucinations of hearing resulted from lesions of the inferior surface of the frontal lobe, but in these cases the olfactory bulbs were directly involved. They also refer to cases of embolism of the artery of the fissure of Sylvius, in which anosmia of the nostril on the side of the lesion occurred. I have failed to find accounts of autopsies in which lesions of the temporal lobe had been followed by olfactory disturbance. I think, however, there can be no reasonable doubt that the hallucinations in my case were connected, in some way, with the morbid

process, and to my mind, it confirms the hypothesis of a participation of the temporal lobes in the sense of smell.

Ferrier, in the first edition of his work, (*Functions of the Brain*, p. 175, et seq.,) concludes, as the results of experiments, that the gyrus hippocampi is the seat of tactile sensibility, destruction of it producing anæsthesia of the opposite side. I have not seen the last edition of his book, but in a recent debate on the localization of the muscular sense, he says:* "As to the cortical localization of the so-called muscular sense, I hold that the centre for this and for all forms of tactile and common sensibility is the falciform lobe," by which I understand him to mean the convolution of the corpus callosum, in addition to that of the hippocampus. So far as I am aware, this view is not shared by any other prominent investigator. The general opinion of those who assent to the doctrine of cerebral localization, is that tactile impressions are perceived in that part of the parietal lobule electrical stimulation of which produces muscular movements—the so-called motor region. The question, therefore, becomes of some interest, whether the case under consideration throws any light on this point.

I think I am entirely safe in saying that there was no very extensive and profound impairment of tactile sensibility. It is also true that the cortex of the region in question was not extensively involved, but the parts lying immediately underneath were affected to such an extent that it can hardly be supposed that its connections remained unimpaired. It seems to me very improbable that there could be so little disturbance of cutaneous sensibility if Ferrier's view as to its centre is correct.

In the following case, symptoms of partial word-deafness were associated with lesion of the first left temporal convolution.

The patient, a Frenchman by birth, aged fifty-eight, was received on the 25th of March, 1885. Before the war he had been a rather prosperous business man in a southern State. The war ruined his business. He was drafted into the Confederate army, taken prisoner, and subsequently enlisted into the Federal army. Since the war he had supported himself by giving lessons in French and German. He had probably been rather a high liver in his more prosperous days, and suffered a good deal from gout, of which he had repeated attacks while a patient in the asylum.

About two months previously to his admission he had an illness, of which no very satisfactory account could be obtained. He

* "*Brain*," Part XXXVII, p. 23.

was stated to have suffered from giddiness, to have been at times almost unconscious and very helpless, and to have lost the power of speech.

At the time of his admission he was still very feeble; could walk a little with the help of a cane. He is stated by Dr. Ward, who received him, to have dragged the right foot at that time, but when he came under my care, about three months later, there was no noticeable difference in his power over the extremities of the two sides. He always walked stiffly, with short steps, but it was not easy to determine just how much of the impairment of locomotion was due to paralysis and how much to stiffness from gout.

At the time of his admission, and for some time afterward, he spoke an almost completely unintelligible jumble of English, French and German words. He often mispronounced words, but this seemed due to want of a correct idea of how they should be pronounced, and not to any paralysis of the vocal organs. He was perfectly well aware that he was not expressing himself properly, and his efforts to find the correct expression were often painful to himself and others. It was also evident that he often failed to understand what was said to him. After repeated trials it would frequently be impossible to make him comprehend a perfectly simply verbal direction or question. The sense of hearing seemed entirely unimpaired. He was emotional and irritable, becoming angry on very slight provocation.

During his residence in the asylum he improved somewhat, both mentally and physically, but there was no essential change in the character of his symptoms. He became strong enough to walk to a considerable distance. He engaged with considerable interest, but no great skill, in playing chess and checkers, and spent a good deal of time in reading—with just how much comprehension of what he read, it was not easy to determine. During the whole time, it was evident that he often had difficulty in comprehending what was said. He gained somewhat in power of oral expression, and during the latter part of his life seldom confused different languages in conversation, replying in that in which he was addressed. His sentences, however, were always very much broken, with numerous repetitions of the same word. During the whole time he wrote pretty frequent letters to a friend, and during the latter part of the time was able to express himself much better in writing than orally. The following letter, bearing date May 11, 1885, will give a better idea of his style of expression than any description:

"Dear friend dearest my love George I pense you to come see you to come sure come be to be sure the evening this evening.

I long to essaye to hear me this week to come to come this week you sure to come me assure to see you come. I hope you be very sure me come this day arrive me possible this sure come sure to come to see dearest my friend now to have you assure us to make be this your day come this week ready your me to write your coffey this week this day if possible.

Make my your happy to hear you sure this evening to come this week.

My love forever sure to come

dear friend

ARTHUR D'A."

This letter, like all that he wrote, was written in a neat and perfectly legible hand, with but few errors in spelling or capitalization. He frequently omitted punctuation marks, but when he used them it was, as a rule, appropriately. He was never able to express himself, in conversation, much better than as above, but the following letter will show that he gained very materially in command of written language:

OCTOBER 6, 1885.

Dear George:

Your letter was received last night. Was glad to have you write, and wish to assure you of my sympathy in your poor state of health. Shall be pleased to have you come and see me if able, if not please write and let me know how you are, for I shall always feel anxious for you when so unwell.

Believe me your best friend as ever,

ARTHUR D'A.

On the 5th of January, 1887, the patient was found in a comatose condition, from which he never rallied. There was no appearance of hemiplegia, as all the extremities moved when the skin was pinched or pricked. He died on the tenth.

At the autopsy, extensive degeneration of the blood vessels of the brain was found; the right vertebral artery was occluded a short distance below its junction with the left to form the basilar, and the left middle cerebral was partially obstructed near its origin. On the upper surface of the first left temporal convolution the cortex was atrophied for a space of about one and one-half inch in length by one and one-half inch in breadth. The lesion was evidently an old one; doubtless there had originally been softening from arterial obstruction, and subsequent absorption of the ne-

crossed tissue. A small, superficial patch of softening was found in the right gyrus supra-marginalis. The interior of the right optic thalamus was mostly transformed into a diffuent mass. The portion of the right nucleus caudatus, immediately opposite the anterior end of the optic thalamus, was shrunken, evidently from atrophy of a patch of old softening, which also affected, to a limited extent, the subjacent portion of the internal capsule.

It is, I presume, hardly necessary for me to enter into argument to prove the connection, in this case, of the affection of speech, and the impairment of power to understand spoken language, with the lesion of the superior temporal convolution. The relation of word-deafness to disease of the left temporal lobe seems to be about as well established as that of aphasia to the third left frontal convolution. Luciani and Seppilli* have collected twenty cases of complete inability to understand spoken language, with preservation of the sense of hearing. In every one of these there was lesion of the first left temporal convolution. In fourteen, the second temporal convolution was also affected, while the largest number of instances in which any other gyrus was involved was six, in the case of the left gyrus supra-marginalis, which, from its contiguity might be expected to be frequently affected in lesions involving the first temporal. The incompleteness of the symptoms in my case is easily understood, in view of the small extent of the diseased portion. The defect of language would be accounted for by the loss of memory of words, which is doubtless dependent upon the same portion of the cortex as their original perception. This has been a prominent symptom in most of the cases which I have seen reported. The superior command of written language would seem to be readily accounted for by the integrity of the visual centres.

The following case is, perhaps, of enough interest to justify quotation, being, so far as my recollection extends, the earliest history of this affection which has come under my notice. It is found in the "*Zoönomia*" of Erasmus Darwin, (4th American edition, vol. II, p. 426.)

"The following curious account of this defect of association of ideas, with audible but not with visible symbols, was sent me by Dr. Darwin, of Shrewsbury.

"The case of an old man lately occurred to me who was superannuated; his hearing and vision were perfect, but he could only

* Functions-Localisation auf der Grosshirnrinde. (German translation,) pp. 205-214.

call up a train of ideas from the latter. When he was told it was nine o'clock, and time for him to eat his breakfast, he repeated the words distinctly, but without understanding them. His servant put a watch into his hand; 'why, William, have I not my breakfast, for it is past nine o'clock,' he would say with expression, that showed he felt what he said. On almost every occasion his servants conversed with him by visible objects, although his hearing was perfect; and when this kind of communication was used, he did not appear impaired in his intellect. This state came on from a stroke of the palsy, and till he and his attendants used this kind of language he was quite childish."

There can, I presume, be little doubt about the character of this case, although the command of language would seem to have been preserved in an unusual degree.

In the following case, beginning with left crural monoplegia, the notes of the autopsy, made at the time, were unfortunately mislaid, and the fact was not discovered until it was too late to fully supply the deficiency from memory. This deficiency, however, is of no special importance so far as the special point illustrated by the case is concerned.

The patient, a colored man, sixty-six years of age, complained, on the 17th of November, 1886, of numbness and weakness of the left leg. This increased rapidly, and when I saw him, at the evening visit, he was unable, when sitting in a chair, to raise the left foot from the floor. There was no paralysis of the facial muscles, and the power of the left hand was but slightly, if at all, impaired. Sensibility in the left lower extremity was evidently diminished, but his mental condition was such that only rough tests could be made. He retained considerable use of the arm until the morning of the 21st, when that also, rather suddenly, became paralyzed. At the first examination, diagnosis was made of lesion of the upper part of the right anterior central (ascending frontal) convolution. It was now supposed that the disease had extended so as to involve the middle portion of the same gyrus. He gradually failed in strength, and had several convulsive seizures. Death occurred on the morning of December 10th. At the autopsy the cerebral arteries were found to be very extensively diseased. A superficial patch of softening was found, involving the upper part of the right anterior central convolution and extending to the paracentral lobule on the internal surface of the hemisphere. Several other spots of softening were found, the precise locations of which I am not now able to state. None of them, however, were in the excitable area of the cortex.

The lesion discovered accounted satisfactorily for the paralysis of the lower extremity. The cortical centre for the arm is usually located lower than the lesion in this case extended, but parallel cases are not wanting. Cases 21, 23 and 25 of Exner's collections seem to have been very similar, clinically as well as anatomically, to this case. I am not, however, able fully to account for the completeness of the paralysis in the upper extremity.

